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Mendelian machinery at work evident. Unwelcome as these conclusions may sound to obscurantists and to those in general who have an antipathy for exact modes of procedure, the necessity for such refined methods here should be obvious. In particular, it will be desirable to examine more intensively those cases which show the characteristics described in Section 5 for balanced lethal factors.

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[1] The full account of this case will appear subsequently, and also an account, by Altenburg and Muller, of the truncate case.

[2] Sturtevant also had performed certain linkage experiments in which beaded was present; these showed that it was well on the other side of sooty from pink.

[3] Since the above has gone to press, a case in *Campanula* which may be due to balanced lethals has been reported by Miss Pellew in the *Journal of Genetics.*

IS DEATH FROM HIGH TEMPERATURE DUE TO THE ACCUMULATION OF ACID IN THE TISSUES?

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I find that there is a converse relation between the rate of oxygen consumption in reef corals and their ability to resist high temperature, those corals which are most readily killed by heat having the highest metabolism (rate of oxygen consumption).

NAME OF CORAL	CONSTANT TEMPERATURE EXPOSURE TO WHICH CAUSES DEATH IN ONE HOUR	RELATIVE RATE OF OXYGEN CONSUMPTION PER GRAM OF LIVING SUBSTANCE IN EACH CORAL
	°C.	
Acropora muricata	34.7	18.7
Orbicella annularis	35.60	6.1
Maeandra areolata	36.80	5.5
Favia fragum	37.05	3.8
Siderastrea radians	38.20	1.0*

* At 28.5°C. 1 gram of living substance of *Siderastrea radians* consumes 0.0256 cc. of oxygen per hour. The oxygen being measured at 0°C. and 760 mm. pressure.

Also, if sea water be super-saturated with carbon dioxide gas, the toxic effect is in the same sequence as that of high temperature. That is to say, those corals which are readily killed by heat are also correspondingly easily killed by H_2CO_3.

This toxic effect of carbon dioxide is not due to its replacing some of the oxygen of the sea water, for I find that corals are remarkably insensitive to a reduction in the oxygen supply, all species except *Acropora* living more than eleven hours in sea water under an air pump which reduced the oxygen to less than 5% of that of normal sea water; and even *Acropora* can withstand six hours of this treatment.

We know, indeed, from the studies of M. Henze that sea anemones use less oxygen the less its concentration in the sea water, and in 1917, J. F. McClendon found that the medusa *Cassiopea* can survive without apparent injury for more than seven hours in the absence of oxygen, and during this time does not give out CO_2. Thus these cœlenterates can temporarily suspend their metabolism for a protracted period if oxygen be absent.

Winterstein's theory that death from heat is due to asphyxiation appears to be refuted by these experiments.

E. N. Harvey, 1911, found that if the sea water be heated the rate of nerve conduction in *Cassiopea* augments in a right line ratio up to a certain point and then rapidly declines just before death ensues.

In 1917, I found that this temperature curve, up to its maximum point, has no time factor. That is to say, the rate at 35°C. is the same whether the medusa be placed at once in 35°C. or warmed slowly for several hours until it arrives at this temperature. Moreover, the normal rate for 29° is regained almost immediately when the medusa is replaced in this normal temperature.

When the rate is declining, due to injuriously high temperature, however, I find that a time factor is involved, the decline becoming more pronounced as the heat is continued. Also, if after this the medusa is replaced in sea water of 29°C., its former rate is much reduced and may never be recovered, although if exposure to the heat was not too long or the heat not too excessive, a slow recovery is usually observed so that after a few hours the rate may again become normal.

It will be recalled that Harvey, 1911, advanced the theory that some enzyme might be destroyed by the excessive heat, and being essential to nerve conduction, its loss caused the rate to decline.

Our experiments however make it seem more probable that some toxic acid substance, possibly lactic acid or H_2CO_3, is formed under the influence of excessive heat. It is easy to see how an acid of this sort might be eliminated and the rate gradually restored when the animal is replaced in normal sea water, whereas if an enzyme were destroyed it might not so readily be replaced.

In any event one or the other of the above mentioned hypotheses seems more in accord with the facts than does Winterstein's asphyxiation theory or the theory that death from heat is due to coagulation of proteid substances.

Death occurs at too low a temperature for coagulation in most if not all proteids; and when killed, the animals are fully relaxed as shown by Harvey. Moreover, coagulated proteins could not readily be eliminated when the animal was restored to water at normal temperature.